sented to demonstrate the successful use of halothane in restoring the blood pressure to normal levels.

The patient, a 28 year old, Negro, male quadriplegic was scheduled for transurethral resection to relieve relative bladder-neck obstruction. Fracture of the cervical spine at the sixth cervical segment, sustained in an accident four years before, resulted in complete sensory loss, and motor loss except for minimal use of the muscles of his right arm. He had a fairly efficient automatic reflex bladder. Three years before he required surgery for removal of bladder stones and a transurethral resection was done at the same time. During the operation, he developed a severe headache and a "stuffed up nose and they had a lot of trouble with my blood pressure." Physical examination on admission was essentially normal, except for the neurological findings already described. (Blood pressure 128/60, pulse 100, hemoglobin 14.4 g., and white blood count 7,000. Urine showed a pseudomonas pyuria.)

The patient was given 0.4 mg. atropine one hour before operation. The surgeon elected to attempt the operation without anesthesia, but the patient was so apprehensive that he was given 75 mg. thiopental and 12 mg. alphaprodine, intravenously. The procedure progressed uneventfully for twenty minutes. The patient’s blood pressure had been stable at 120/80, then rose precipitously to 200/90, and his pulse slowed to 60. He became restless and complained of headache and a "stuffy nose." In spite of 150 mg. thiopental and 12 mg. alphaprodine intravenously, his blood pressure continued to rise. He complained bitterly of the pain in his head. Halothane (2 per cent) with 50 per cent N₂O in O₂ was started. The blood pressure fell immediately to 170/90 and within five minutes both pulse and blood pressure returned to initial levels. The concentration of halothane was reduced to 0.5 per cent. The surgeons continued the operation without interruption and were able to distend the bladder without recurrence of the autonomic reflex. The patient awoke immediately at the conclusion of operation with no complaints. His postoperative course was entirely uneventful.

General anesthesia is often desirable to avoid reflex muscular movements and spasm even though the paraplegia may have complete sensory loss. It also seems preferable to spinal anesthesia in the presence of spinal cord injury. The severity of the hypertensive crises has, however, created a dilemma. Halothane may, therefore, be useful in the paraplegic since it appears to provide protection against hypertensive crises encountered with other general anesthetics.

References

CORRESPONDENCE

Controlled Acid-Base Status with Cardiopulmonary Bypass and Hypothermia

To the Editor.—I was very interested in the article, "Controlled Acid-Base Status with Cardiopulmonary Bypass and Hypothermia" by Drs. Carson and Morris (Anesthesiology 23: 618, 1962). On page 620 it is stated, "Since the nomogram is designed for blood at 37°–38° C. values for pH and Pco₂ as measured at 37° C. and uncorrected for temperature are used to find the base excess or deficit. It is thought that this value should be valid for blood from hypothermic subjects since base excess or deficit does not change with anaerobic change in temperature."

I believe the assumption that base excess or deficit does not change with anaerobic change in temperature is erroneous and may lead to a misinterpretation of acid-base data. Stadie and Martin have shown that the nor-
normal buffer line of blood is shifted .022 pH units in the alkaline direction for each 1° C. fall in temperature. The assumption by Carson and Morris will, therefore, lead to serious errors in the assessment of the acid-base status of either hypo- or hypertermic patients.

One way of assessing the buffer state of a hypothermic patient is by means of the pH-bicarbonate diagram with $P_{CO_2}$ isobars as designed by Henderson. Such a diagram is shown above and will be used to determine the acid-base status of a hypothetical patient, to illustrate the errors incurred by using Carson and Morris’ method.

Example: Arterial blood is drawn from an anesthetized subject at 37° C, and the serum $pH$ and $P_{CO_2}$ are determined with suitable electrodes maintained at 37° C., indicating a $pH$ of 7.4 and a $P_{CO_2}$ of 40 mm. of mercury. These points are plotted above as point A. These values indicate that a serum bicarbonate concentration of 25 mmoles/liter is present. The patient is then cooled until esophageal temperature is 28° C. Arterial blood is again withdrawn and analyzed in electrodes maintained at 37° C.; $pH$ and $P_{CO_2}$ readings of 7.493 and 40 mm. of mercury are obtained. These values, uncorrected for the difference between electrode and body temperature, are plotted as point B on the diagram, indicating that a serum bicarbonate concentration of 31.2 mmoles/liter is present. The vertical distance between point B and the normal buffer line at 37° C. indicates a base excess of 8.8 mmol/liter. However, the hypothermic $pH$ and $P_{CO_2}$ should be corrected to body temperature ($pH$ 7.625, $P_{CO_2}$ 27 mm. of mercury) and referred to a buffer line and $P_{CO_2}$ isobar which have also been corrected for body temperature. These values are plotted as point C indicating that in reality a base deficit of 3.1 mmol/liter has occurred and that this patient is in mild metabolic acidosis.

Use of the Astrup nomogram as Carson and Morris have done will always result in an over-estimation of buffer base concentration. However, this may not interfere with the qualitative conclusions of this study; that metabolic acidosis is less profound in hypertermic patients on cardiopulmonary bypass.
when arterial blood pH is lowered by the addition of carbon dioxide to the oxygenator respiratory mixture. Recalculation of their data is not possible since $P_{CO_2}$ values were not included.

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References

Effect of Cyclopropane and Halothane on the Blood Volume

To the Editor.—I read with interest and concern the article entitled: "The Effect of Cyclopropane and Halothane on the Blood Volume in Man" by Ernest Grable and co-workers, which appeared in Anesthesiology 23: 828, 1962. There are several points I wish to bring out which may be of interest to readers of the above mentioned article.


(2) It is most advisable for anyone who wishes to measure blood volume and in particular when interpreting plasma volume, to read and understand what is being measured. One actually tries to interpret the protein distribution space as plasma volume—a noteworthy misconception.


(3) I wish to congratulate the authors on having mastered the use of iodine$^{125}$ albumin. Over a period of six months we tried to use this radioactive isotope of iodine, generously supplied by E. R. Squibb & Sons, and after many in vitro trials and repeated consultations with colleagues versed in this subject, we have come to the conclusion that $I^{125}$, a very weak gamma emitter (35.4 kev.), is absorbed by chromium$^{51}$ red cells absorb part of the energy—even protein concentration in the solution alters the accuracy in measuring $I^{125}$ concentrations and, therefore, a word of caution is in order on the use of this tracer.

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